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Review

The mutagenic hazards of settled house dust: a review

Rebecca M. Maertens, Jennifer Bailey, Paul A. White*

Mutagenesis Section, Safe Environments Program, Health Canada, Tunney's Pasture 0803A, Ottawa, Ont., Canada K1A 0L2

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Abstract

Given the large proportion of time people spend indoors, the potential health risks posed by chemical contaminants in the indoor environment are of concern. Research suggests that settled house dust (SHD) may be a significant source for indoor exposure to hazardous substances including polycyclic aromatic hydrocarbons (PAHs). Here, we summarize the literature on the mutagenic hazards of SHD and the presence of PAHs in dust. We assess the extent to which PAHs are estimated to contribute to the mutagenicity of SHD, and evaluate the carcinogenic risks associated with exposures to PAHs in SHD. Research demonstrates that SHD has a *Salmonella* TA98 mutagenic potency of 1000–7000 revertants/g, and contains between 0.5 and 500 μg/g of PAHs. Although they only account for a small proportion of the variability, analyses of pooled datasets suggest that cigarette smoking and an urban location contribute to higher levels of PAHs. Despite their presence, our calculations show that PAHs likely account for less than 25% of the overall mutagenic potency of dust. Nevertheless, carcinogenic PAHs in dust can pose potential health risks, particularly for children who play and crawl on dusty floors, and exhibit hand-to-mouth behaviour. Risk assessment calculations performed in this study reveal that the excess cancer risks from non-dietary ingestion of carcinogenic PAHs in SHD by preschool aged children is generally in the range of what is considered acceptable (1×10^{-6} to 2×10^{-6}). Substantially elevated risk estimates in the range 1.5×10^{-4} to 2.5×10^{-4} correspond only to situations where the PAH content is at or beyond the 95th percentile, and the risk estimates are adjusted for enhanced susceptibility at early life stages. Analyses of SHD and its contaminants provide an indication of indoor pollution and present important information for human exposure assessments.

Keywords: Dust; Mutagenicity; PAH; Risk assessment; Cancer

Contents

* Corresponding author. Tel.: +1 613 941 7373; fax: +1 613 941 8530.
E-mail address: paul_white@hc-sc.gc.ca (P.A. White).

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1. Introduction

Much attention has been placed on researching, monitoring and regulating air pollution in the outdoor environment. As a result, there exists a general misconception that air pollution by chemical contaminants is an outdoor phenomenon. In reality, numerous studies have noted that indoor air can be many times more contaminated than outdoor air [1]. Moreover, people spend the majority of their time indoors. For example, Canadians spend as much as 70% of their time at home and up to 90% of their time indoors [2]. These percentages are easily exceeded for mothers, children, the elderly and the infirm. As a result, the health risks posed by contaminants in the indoor environment are of significant concern, and the potential hazards of indoor pollutants are now being more widely acknowledged. For instance, organizations such as the United States Environmental Protection Agency (US EPA) have recently ranked indoor pollution as a high priority risk to human health [3].

Pollutants in the indoor environment can include radiation (e.g., radon gas), biological contaminants (e.g., bacteria, molds, viruses, dust mites), chemical contaminants (e.g., pesticides, metals, flame retardants, plasticizers), combustion products (e.g., environmental tobacco smoke, carbon monoxide, nitrogen dioxide) and others [4]. Many of these contaminants adsorb to particulate matter suspended in indoor air that later settles out as house dust.

Research investigating human exposures to priority pollutants have suggested that settled house dust (SHD) may be a significant source for indoor exposures [5]. Exposure to these pollutants in the indoor environment has been associated with numerous adverse health effects including allergenic and immune system effects, respiratory effects, cardiovascular and nervous system effects, irritating effects of the skin and mucous membranes, cancer and reproductive effects [6]. Exposure to dust and its associated contaminant load may be of particular concern for children who tend to play or crawl on the floor and place objects in their mouths that have been in intimate contact with dusty floors or carpets [7].

Studies investigating exposure to toxic contaminants in SHD have often focussed on lead [8–13] and pesticides [7,14–18]. However, combustion products such as the polycyclic aromatic hydrocarbons (PAHs) have also been detected in dust, and these substances may pose additional health concerns [19–39]. PAHs are ubiquitous in the indoor and outdoor environment, and a number of these compounds have been classified as mutagens and/or possible or probable human carcinogens [40].

Although several studies have investigated the PAH composition of SHD, only one published study [41] has investigated the mutagenic hazards of SHD. As a

result, there is a paucity of information on the mutagenic hazards of indoor dust. Given the large proportion of time spent indoors and the potential for enhanced risks in children, the hazards associated with exposure to indoor dust warrant further investigation. The objectives of this review are: (1) to review the limited data on the mutagenicity of SHD, (2) to compile published data on PAH levels in SHD, and analyze relationships between these levels and various attributes of the households (e.g., location, presence of smokers, type of flooring), (3) to assess the potential contributions of frequently measured PAHs to the overall mutagenic hazards of SHD, and (4) to estimate the carcinogenic risks associated with exposure to PAHs in SHD.

1.1. Composition of house dust

The US EPA defines house dust as "a complex mixture of biologically-derived material (animal dander, fungal spores, etc.), particulate matter deposited from the indoor aerosols, and soil particles brought in by foot traffic ... The indoor abundance depends on the interplay of deposition from the airborne state, re-suspension due to activities, direct accumulation and infiltration" [42]. The precise composition of a house dust sample is a function of numerous factors including environmental and seasonal factors, ventilation and air filtration, homeowner activities, and indoor and outdoor source activities. The penetration of outdoor particles into the indoor environment has been shown to be a significant source of indoor particles [43–46]. In the outdoor environment, natural sources of dust particles include pollen, soil, forest fire emissions and volcanic debris. Anthropogenic sources of outdoor dust particles include fossil fuel combustion (e.g., coal, oil), wood combustion, waste incineration, and a variety of industrial processes (e.g., iron founding, construction). In the indoor environment, dust sources include skin, hair, mites, fibres from clothing and furnishings, cooking emissions, heating emissions and cigarette smoke [47]. This variety of indoor and outdoor sources yields a complex matrix that can be extremely heterogeneous in nature with temporal and spatial variability in particle size, particle shape, particle composition, and contaminant concentration. Consequently, the composition of SHD can differ considerably between rooms of a given house, as well as between houses, and among geographic locations in a study area [20].

Most dust particles range from micrometers to millimetres in size and are generally classified as either fine or coarse particles. Although no standard exists, a common practice is to define fine particles as those less than 2.5 μm, while coarse particles are those greater than 2.5 μm [47]. Dust particle size is of particular importance as it influences the deposition and re-suspension of dust in the indoor environment. Particles greater than 30 μm tend to fall and form SHD [47], while particles less than 30 μm tend to remain airborne and only constitute approximately 10% of SHD [31,48,49]. The settling and re-suspension of dust is readily influenced by air flow patterns and activities taking place in the sampling area [50,51].

The physical–chemical characteristics and composition of house dust plays an important role in determining the types of contaminants that are associated with dust particles. The adsorption and adherence of chemical contaminants to particulate material depends on the type and size of the particles as well as the surface texture, polarity and lipophilicity [19]. Studies have revealed the presence of many chemical contaminants adsorbed to dust particles including: pesticides, smoke residues, PAHs, PCBs, flame retardants, plasticizers, heavy metals and asbestos [19–22]. Equilibrium concentrations on dust particles generally far exceed those in the gaseous portion of indoor air [52], thus dust and its associated fine particulate matter tends to become a sink for semi-volatile organic compounds [19]. Furthermore, these compounds have the potential to persist and accumulate in indoor dust, as they are not subjected to the same degradation processes that occur outdoors. Compounds associated with indoor dust particles are protected from sunlight, fluctuations in temperature and humidity, high rates of microbial degradation, and the overall effects of weathering [53].

Some of the general characteristics of SHD are presented in Table 1. It should be noted however, that due to the complex nature of SHD and the numerous factors that influence its composition, actual values for specific dust characteristics (i.e., deposition rate, particle size distribution and loss on ignition) may vary considerably from the values shown in Table 1 depending on the location that is being sampled. For a

Table 1
General characteristics of settled house dust (SHD)

Characteristic	Typical values	References
Loading	0.6–1.3 g/m^2	[18,50,97]
Deposition rate	0.0022–0.08 g/m^2 per day	[56,98,99]
Particle size distribution	>125 μm (40%) 50–125 μm (41%) 25–50 μm (18.3%) <10 μm (0.6%)	[49]
	63 μm–2 mm (37.2%) <63 μm (23.1%)	[100]
Loss on ignition[a]	63 μm–2 mm (38.6%) <63 μm (58.6%)	[100]

[a] A measure of organic carbon content.

more detailed overview of the sources and properties of SHD, the reader is referred to the recent publication by Morawska and Salthammer [47].

1.2. Collection of settled house dust samples

Researchers investigating dust contamination have devised a number of passive and active dust sampling techniques. Passive techniques may involve setting out stationary "dust fall" jars or non-electrostatic plates and simply letting dust accumulate for a given period of time. Active sampling techniques can include: surface wiping, press sampling, sweeping, or vacuuming. Each of these methods has been devised to measure specific parameters such as the total dust loading or dust available for dermal adsorption. No one sampling method can collect dust equally well from all surfaces, and the optimal collection method will depend on the surface to be sampled and the goal of the study. A comprehensive review of the various sampling techniques is provided by Lioy et al. [20].

In an effort to obtain the most reliable information with the highest possible reproducibility, two standard methods for sampling SHD have been established; one by the American Society for Testing and Materials (ASTM), and the other by the German Association of Engineers (VDI). The ASTM method D 5438-00 makes use of the High Volume Small Surface Sampler (HVS3), a modified vacuum cleaner that collects particles greater than 5 μm using various cyclones [54]. The VDI 4300 Part 8 guideline describes methods for a number of sampling techniques (e.g., commercial vacuum cleaners, surface wipes, deposition collection) in order to optimize sampling to the specific situation [55]. This guideline also distinguishes between "old dust" which is dust of unknown age, and "new dust" which is generally 1–2 weeks old. The collection methods employed in many published studies do not adhere to any rigid standards. This introduces unfortunate variability (e.g., in particle size distribution), which complicates cross-study data analysis and interpretation.

Reviews by Butte and Heinzow [19], Roberts and Dickey [52], and Roberts et al. [5] provide an overview of dust sampling studies to date. They also summarize the occurrence of various chemical contaminants in dust and assess potential exposure rates.

1.3. Exposure to settled house dust

Exposure to SHD and associated contaminants may occur via dermal adsorption, inhalation, and non-dietary ingestion. Dermal absorption of dust may occur following contact with dust that has settled on furniture, floors or other objects. Dust particles less than 100–200 μm are most effectively retained by the skin [31]. It is estimated that approximately 28 mg of SHD per day adsorb to children's hands, while 51 mg adsorb to the hands of adults [56]. In non-occupational settings, this route of exposure is thought to be less significant than inhalation and non-dietary ingestion [29].

Inhalation of dust can occur when dust is suspended or re-suspended by activities such as vacuuming, cleaning, playing, or simply walking through a room [50]. It is estimated that young children inhale between 0.15 and 0.34 mg of dust per day, while adults inhale approximately 0.81 mg per day [56]. Inhaled dust particles greater than 10 μm are generally trapped by the nose, throat or upper respiratory tract, whereas particles less than 2.5 μm have the ability to penetrate deep into the respiratory system where they are less likely to be eliminated [47]. These finer particles, which often contain higher levels of PAHs [31], likely pose a toxic hazard to exposed individuals.

Non-dietary ingestion of SHD generally occurs through accidental ingestion of particles that have adhered to food or skin. This route of exposure is thought to be of particular concern for children who frequently put their hands, toys and other objects into

their mouths [7]. It is estimated that young children ingest between 50 and 100 mg of dust per day compared to adults who ingest an estimated 0.56 mg per day [56]. A small percentage of children are known to exhibit pica behaviour, which involves the intentional eating of non-food items. These children may ingest up to 10 g of soil and dust per day [57].

1.4. Mutagenicity of house dust

Other than our current work examining SHD samples from homes in the Ottawa area, to our knowledge only one study has investigated the mutagenic hazards of SHD. In their study of 29 houses in Washington state, Roberts et al. [41] examined the mutagenicity of dust collected from homes in high and low pollution areas. A microtiter *Escherichia coli* K-12 DNA repair assay and the *Salmonella* mutagenicity assay (TA98 only) were used to assess the genotoxicity of the dust extracts.

Statistically significant increases were noted for both assays. Specifically, 20 out of 29 samples gave statistically significant positive responses in the DNA repair assay. Ten out of 29 samples yielded significantly elevated levels of *Salmonella* mutagenicity in the absence of metabolic activation, and 5 out of 29 samples showed significantly elevated levels of *Salmonella* mutagenicity in the presence of metabolic activation (S9). *Salmonella* (TA98) mutagenic potency values ranged from 1190 to 6570 revertants/g without S9 and from 1340 to 4180 revertants/g with S9. Eight of the dust extracts produced elevated responses for both the DNA repair assay and the *Salmonella* mutagenicity assay. In addition, both tests revealed an increase in mutagenic activity with decreasing particle size.

Roberts et al. also examined correlations between the mutagenicity of the dust samples and information contained in the corresponding homeowner surveys. The authors found a statistically significant correlation between the age of the carpet and the magnitude of the *Salmonella* mutagenicity with metabolic activation. They also found a significant correlation between vehicle traffic density and the *Salmonella* mutagenicity without metabolic activation. The latter relationship suggests that direct-acting mutagenic combustion by-products such as nitro-substituted PAHs produced outdoors (e.g., diesel emissions) may be entering the indoor environment. The study did not investigate the chemical composition of the dust. Hence, no correlations could be made between the level of mutagenicity and the concentration of any mutagenic compounds present in the dust.

Preliminary *Salmonella* mutagenicity analyses of SHD collected from 65 houses in the Ottawa area confirm mutagenic potency values with TA98 range from 780 to 3678 revertants/g ($N = 13$ positive results) without S9, and 2299 to 7213 revertants/g ($N = 23$ positive results) with S9 activation (Maertens, unpublished data). These values are similar to the values published in the study by Roberts et al., and it appears that the *Salmonella* TA98 mutagenic activity of SHD extracts tends to be in the 1000–7000 revertants/g range. Comparisons between the mutagenic potency of indoor dust and other particulate matrices reveals that settled dust tends to be more mutagenic than most outdoor soils, including those collected from contaminated industrial sites, but less mutagenic than suspended particulate matter collected from either indoor or outdoor air (Table 2). Geometric mean mutagenic potency values for contaminated soils from industrial sites tend to be in the 1000 revertants/g range, although individual values for heavily contaminated soils can yield 10^5 revertants/g [58]. Although the potency of suspended particulate material collected in both indoor and outdoor environments can vary a great deal, organic extracts of these samples often yield potency values greater than 10^5 TA98 revertants/g [59]. This relative ranking of mutagenic potency seems reasonable since settled dust contains deposited particulates from both indoor and outdoor air, as well as tracked-in soil particles [50]. The relatively low mutagenic potency of SHD in comparison to suspended particles in indoor or outdoor air is likely due to the dilution of SHD with large particles of inert material and textile fibers that are non-mutagenic. In a similar fashion, the lower levels of mutagenic potency of soil particles is almost certainly accounted for, at least in part, by the presence of large amounts of inert material of geological origin.

2. Sources of house dust mutagenicity: PAHs

There are a number of substances that could potentially contribute to the mutagenicity of dust.

Table 2
Salmonella TA98 mutagenic potency[a] of dust, indoor air, outdoor air and outdoor soil

Media	Sampling location	Areal/volumetric dust concentrations[b]	Revertants/g, −S9	Revertants/g, +S9	Reference
Settled dust	29 homes in high and low pollution areas	1,900,000 ± 300,000 μg/m^2	1190–6570[c]	1340–4180[d]	[41]
Settled dust	Preliminary results for Ottawa homes	NA	780–3678	2299–7213	Maertens, unpublished data
Indoor air	39 Rural homes	37–210 μg/m^3	40,000–60,000	240,000–550,000	[101]
	One home in a residential area	NA	70,000–460,000	130,000–370,000	[102]
	24 Rural homes	36–59 μg/m^3	120,000–260,000	280,000–450,000	[103]
	Four urban homes	50–110 μg/m^3	12,000–78,000	14,000–187,000	[104]
	One rural home	30–140 μg/m^3	6000–36,000	17,000–49,000	
Outdoor air	One industrial location	5.13–13.73 μg/m^3		26,000–87,330	[105]
	Two urban locations	3.97–5.75 μg/m^3		17,920–50,910	
	One industrial location	68.5 μg/m^3	520,000	577,000	[106]
	One industrial location	5.3–15.8 μg/m^3	1,000,000–1,537,974	867,924–1,649,425	[107]
	One agricultural location	3.6–8.2 μg/m^3	638,889–2,097,560	750,000–1,329,268	
Soil	Heavily contaminated sites	NA	770 ± 180	950 ± 170	[58]
	Urban/industrial sites	NA	430 ± 10	470 ± 50	[58]
	Remote/rural sites	NA	57 ± 6	60 ± 5	[58]

[a] Defined as the initial slope of the concentration–response curve (see Bernstein et al. [108] or similar).
[b] Mean value or range where available.
[c] Range for 14 positive samples.
[d] Range for 15 positive samples.

These could include a host of organic and inorganic compounds commonly associated with a variety of industrial products (e.g., textiles, paints, furniture) such as hexavalent chromium, nickel compounds, styrene, tetrachloroethylene, benzidine and vinyl chloride [60–63].

Of particular interest, one of the groups of chemicals suspected of contributing to the mutagenic activity of dust is the polycyclic aromatic hydrocarbons and related compounds (e.g., nitro-arenes, heterocyclics) [40]. PAHs, several of which are known mutagens, are products of incomplete combustion and are ubiquitous in indoor and outdoor environments. Their involvement in determining the mutagenicity of indoor dust is consistent with the aforementioned relationship between mutagenicity and traffic density, as exhaust fumes are a major source of PAHs [64]. Indoor sources of PAHs include cooking [65,66], heating [67], smoking [68], wood burning [69], candle burning [70] and incense burning [65,71]. Outdoor sources include vehicle exhaust [64] and industrial processes such as aluminium smelting, coke production, and petroleum refining [72]. To a lesser extent, environmental PAHs can also be petrogenic in origin [73]. Table 3 summarizes the mutagenic and carcinogenic properties of several PAHs including those listed as priority substances by the US EPA [74].

Most PAHs, particularly those that are known to be mutagenic and carcinogenic, have low vapour pressure, low water solubility, and high octanol–water partition coefficients (K_{ow}) [75]. Compounds with low vapour pressure and high K_{ow} are expected to be adsorbed to particulate material. Table 4 includes a summary of the physical–chemical properties of several PAHs, and the calculated fraction of each PAH that would be expected to be adsorbed to indoor particulate matter at 25 °C. This value, calculated using the level I fugacity models of MacKay [75], reveals that, for PAHs with log K_{ow} values greater than 4.0 and vapour pressure values less than 1.0, almost all of the PAH will be adsorbed to particulate (aerosol) material. However, it should be noted that this calculation is based on a simplified indoor system composed only of air, water (humidity) and particulate material. Thus, it could not account for PAHs adsorbed to indoor surfaces (e.g., walls, furniture), since the fugacity capacities of these indoor surfaces are not known.

Table 3
Mutagenicity and carcinogenicity of selected PAHs targeted for analysis in published studies of SHD

PAH	CAS registry number	Mutagenicity[a]	Carcinogenicity (IARC)[b]	Carcinogenicity (IRIS)[c]	Toxic under CEPA 11(c)[d]
Acenaphthene	83-32-9	0	Not assessed	Not assessed	Not assessed
Acenaphthylene	208-96-8	No data	Not assessed	D	Not assessed
Anthracene	120-12-7	1[e]	3	D	Not assessed
Benz[*a*]anthracene	56-55-3	1, 2, 3	2A	B2	Not assessed
Benzo[*a*]pyrene	50-32-8	1, 2, 3	2A	B2	Yes
Benzo[*e*]pyrene	192-97-2	1, 2	3	Not assessed	Not assessed
Benzo[*b*,*k*]fluoranthene	205-99-2, 207-08-9	1	2B	B2	Yes
Benzo[*g*,*h*,*i*]perylene	191-24-2	1	3	D	Not assessed
Chrysene	218-01-9	1, 2	3	B2	Not assessed
Coronene	191-07-1	1	3	Not assessed	Not assessed
Cyclopenta[*c*,*d*]pyrene	27208-37-3	1, 2	3	Not assessed	Not assessed
Dibenz[*a*,*h*]anthracene	53-70-3	1, 2	2A	B2	Not assessed
Fluoranthene	206-44-0	1, 2	3	D	Not assessed
Fluorene	86-73-7	0	3	D	Not assessed
Indeno[1,2,3-*c*,*d*]pyrene	193-39-5	0	2B	B2	Yes
Naphthalene	91-20-3	2[f]	2B[g]	D	Not assessed
Phenanthrene	85-01-8	1, 2	3	D	Not assessed
Pyrene	129-00-0	1, 2	3	D	Not assessed

[a] Based on information from IARC [40], the genetic activity profile database (GAP2000) [109] and the National Toxicology Program [110]. 0, no evidence of mutagenicity; 1, mutagenic in bacterial and/or fungal/yeast cells in vitro; 2, mutagenic in plants or animal cells in vitro; 3, mutagenic in the *Drosophila melanogaster* somatic mutation and recombination test, and/or sex-linked recessive lethal test, and/or transgenic rodent assays, and/or rodent dominant lethal test.

[b] Based on information from IARC. IARC classification: 1, carcinogenic to humans; 2A, probably carcinogenic to humans; 2B, possibly carcinogenic to humans; 3, inadequate or limited evidence of carcinogenicity in experimental animals [40].

[c] Based on information from the Integrated Risk Information System. US EPA classification: A, human carcinogen; B1, probable human carcinogen (limited human data); B2, probable human carcinogen (primarily on the basis of animal data); C, possible human carcinogen; D, not classifiable as to human carcinogenicity (inadequate or no evidence); E, non-carcinogen [89].

[d] May constitute a danger in Canada to human life or health, as defined under paragraph 11(c) of the Canadian Environmental Protection Act [72].

[e] IARC [40] noted that anthracene failed to induce mutations in bacteria or yeast, and did not induce cytogenetic effect in vitro or in vivo. A single, subsequent publication [111] noted that anthracene can induce mutations in *Salmonella* TA100 in the presence of rat liver or hamster liver S9 (10% v/v in activation mixture).

[f] The NTP Executive Committee Working Group [90] indicates that the majority of genotoxicity tests have shown that naphthalene is not genotoxic in most in vitro assays and cannot induce mutations in bacteria. An earlier IARC publication [60] noted that naphthalene showed clastogenic activity in cultured CHO cells.

[g] IARC [60] and NTP [112], respectively, noted that there is *sufficient evidence* to support the carcinogenic activity of naphthalene in experimental animals, and *clear evidence* of carcinogenic activity in rats. However, the 2002 report of the NTP Executive Committee Working Group [90] could not arrive at a consensus regarding the carcinogenicity of naphthalene. The US EPA [89] stated that naphthalene is not classifiable with respect to its carcinogenic activity.

Only a small number of studies have examined the PAH content of SHD. Table 5 summarizes several of the important published findings. In order to gain additional insight, we compiled a complete dataset containing all available published data on PAHs in SHD. Analyses of the collected data focussed on three issues: the relationship(s) between the PAH content of SHD and various attributes of the home (e.g., location, income, smoking habits), the degree to which commonly measured PAHs can account for the measured mutagenic potency of household dust, and the potential cancer risks posed to preschool children exposed to SHD contaminated with carcinogenic PAHs.

2.1. Collection of published house dust PAH data

PAH composition data were collected from 18 publications including several peer-reviewed journal articles and government reports. In cases where

Table 4
Physical–chemical properties of several PAHs[a]

Compound	Vapour pressure at 25 °C (Pa)	log K_{ow}	Fraction adsorbed to household dust at 25 °C (%)[b]
Acenaphthene	8.93E−01	3.55	98.88
Acenaphthylene	3.78E−01	4.03	97.40
Anthracene	8.31E−04	4.54	99.96
Benz[*a*]anthracene	4.10E−06	5.91	99.99
Benzo[*a*]pyrene	7.00E−07	6.50	100.0
Benzo[*e*]pyrene	7.32E−07	6.44	100.0
Benzo[*b*]fluoranthene	6.67E−05	6.50	99.99
Benzo[*k*]fluoranthene	6.70E−05	6.20	99.99
Benzo[*g*,*h*,*i*]perylene	1.39E−08	7.10	100.0
Chrysene	4.00E−06	5.91	99.99
Coronene	1.95E−10	7.64	99.99
Cyclopenta[*c*,*d*]pyrene	NA[c]	NA	NA
Dibenz[*a*,*h*]anthracene	1.30E−08	6.20	100.0
Fluoranthene	6.42E−03	5.22	99.97
Fluorene	9.46E−02	4.18	99.56
Indeno[1,2,3-*c*,*d*]pyrene	NA	NA	NA
Naphthalene	1.08E+01	3.36	81.71
Phenanthrene	1.61E−02	4.57	99.95
Pyrene	6.00E−04	5.18	99.99

[a] Physical–chemical properties obtained from MacKay et al. [113].
[b] Level I fugacity calculations assuming a home has a volume of 295 m^3, containing 3.4 L of water (i.e., 50% relative humidity) and 8.3 L of particulate material ($\rho = 1172.1 \pm 359.5$ kg/m^3).
[c] Data not available.

published data were difficult to locate, study authors were contacted and reports were acquired directly from the author. Only studies that provided the concentration (μg/g) of PAHs in SHD, as opposed to surface loading (g/m^2), were included. All PAH concentrations in dust were converted to μg/g. The 18 studies contained a combined total of 132 observations that are summarized in Appendix A.

The majority (122) of the 132 observations recorded in Appendix A reflect the results of analyses conducted on samples collected from a single location. The remaining ten observations represent studies that only provided mean or median values for a series of locations. Forty-eight percent of the 132 observations are from urban areas, 15% are from rural areas, and 1% from suburban areas. The remaining observations (36%) are from locations that were not fully described. Most of the observations are from sites where smokers were not present (47%). Fewer observations were collected from sites with smokers (17%), and several studies did not provide information on the smoking habits of the inhabitants (36%). Information on the socio-economic status of the sampled households was not available for much of the dataset (59%). Where

Table 5
Factors associated with PAH composition of SHD

Study area	Variables investigated	Conclusion	References
Ohio, North Carolina	Track-in soil	PAH concentrations in SHD are greater than that in outdoor soil	[29,36]
North Carolina, Minnesota	Air (indoor and outdoor)	PAH concentrations in SHD are correlated with PAH concentrations in both indoor and outdoor air	[114,115]
Texas	Season	PAH concentrations in SHD are higher in the spring than in the summer	[33]
North Carolina	Location	PAH concentrations in SHD from urban areas are higher than from rural areas	[29]
North Carolina	Income	The differences in PAH concentrations between SHD from low-income and middle-income houses are small	[23]
Ohio	Smoking	Smoking is not the primary determinant of PAH levels in SHD	[28]

this information was provided, most of the samples came from low-income areas (40%), while a small number came from medium-income households (1%).

Unfortunately, few of the studies provided detailed information about the methods employed for sample collection, processing, and analysis. The majority of the SHD samples (69%) were collected using the High Volume Small Surface Sampler (HVS3). A smaller number of samples were collected using household vacuum cleaners (4%), and a smaller number still were collected using a combination of sampling techniques (1%) or unidentified methods (26%). The type of surface sampled included carpet only (42%), a combination of surfaces (21%), or unspecified surfaces (37%). In all cases, the dust particles selected for study were less than 150 μm in diameter, except for one study [30] in which the particle size was not indicated. The majority of the SHD samples were extracted using hexane (75%) or diethyl ether in hexane (23%). One study [38] used acetone and cyclohexane, and one study [30] did not specify which extraction solvent was used. Where analytical instrumentation was specified, all studies employed gas chromatography/mass spectrometry for identification and quantification of PAHs. Eighteen PAHs were selected for analysis across the studies. However, the number and identity of the PAHs examined differed across the studies. The most commonly studied PAHs were benzo[*a*]pyrene and benz[*a*]anthracene, compounds which are also among the most mutagenic and carcinogenic (see Table 3).

2.2. Analysis of the collected PAH data

All analyses were performed using the SAS system version 8.02 for Windows [76]. Analysis of variance (ANOVA) was employed to investigate relationships between PAH concentration values (μg/g) and various site attributes (e.g., urban, low-income). Following the notation of Gujarati [77], the general model $Y_i = \alpha_1 + \alpha_2(D_2) + \alpha_3(D_3) + \alpha_n(D_n) + \mu_i$ was fit to the data. Y_i is the observed PAH concentration for observation i, and D_2 through D_n are dichotomous variables that indicate membership of observation i in a given group (e.g., urban sites, low-income sites). D_2 through D_n are set to 1 when the condition of group membership is satisfied and 0 when the condition is not satisfied. Where necessary, the data were log transformed to meet the normality assumptions of ANOVA. The residual error term μ_i was assumed to be independent and normally distributed. Normality was assessed using the Shapiro-Wilk statistic and visual examination of a normal probability plot [78]. The absolute value of the residual error values (μ_i) was used to detect outliers and identify data entry errors. To identify significant outliers, externally studentized residuals (di^*) were calculated for each validated residual error value [79]. All analyses were conducted for total PAHs (i.e., sum of the 18 targeted PAHs), the total low molecular weight PAHs (i.e., those having two or three fused rings), the total high molecular weight PAHs (i.e., those with four or more fused rings) and the total carcinogenic PAHs (i.e., only those PAHs defined by the US EPA as B2 carcinogens).

2.3. Results and discussion

Examination of the raw data (Appendix A) indicates that the PAH content of SHD is extremely variable. Concentrations spanned up to four orders of magnitude for a single PAH, and up to five orders of magnitude across different PAHs. The sum of the reported PAHs for each observation (i.e., total PAH) ranged from approximately 0.5–500 μg/g. The minimum, maximum and mean PAH concentrations are summarized in Table 6. Overall, the PAHs that occurred in the lowest concentrations were acenaphthene, acenaphthylene and cyclopenta[*c*,*d*]pyrene. The low concentrations are likely in part due to the volatile and reactive nature of these PAHs [39]. The PAHs that occurred in the highest concentrations are benzo[*b*,*k*]fluoranthene, fluoranthene and pyrene. Mixtures of PAHs have been shown to have a relatively high abundance of pyrene [80]. Moreover, PAHs with molecular weights of 202, such as fluoranthene and pyrene, can be present in both a gaseous and particle-adsorbed state at room temperature, while PAHs with molecular weights greater than 228, such as benzo[*b*,*k*]fluoranthene, are predominantly associated with particulate matter [80]. This pattern is also consistent with the data shown in Table 4. Compounds such as acenaphthene, acenaphthylene have relatively high vapour pressure values (i.e., ~0.3 Pa) and low K_{ow} values (i.e., 10^4 range), whereas compounds such as benzo[*b*]fluoranthene, benzo[*k*]-fluoranthene and pyrene have far lower vapour

Table 6
Minimum, maximum and mean PAH concentration values in SHD from 18 published studies

PAH	*N*	Minimum (μg/g)	Maximum (μg/g)	Arithemtic mean (μg/g)	S.E.M.[a]	Geometric mean (μg/g)
Acenaphthene	115	0.001	1.900	0.115	0.029	0.032
Acenaphthylene	113	0.001	0.520	0.063	0.008	0.026
Anthracene	125	0.005	5.800	0.284	0.070	0.065
Benz[*a*]anthracene	130	0.017	40.000	1.476	0.421	0.241
Benzo[*a*]pyrene	131	0.015	54.000	2.110	0.597	0.285
Benzo[*e*]pyrene	122	0.015	41.000	1.733	0.503	0.286
Benzo[*b*,*k*]fluoranthene	127	0.030	108.000	4.005	1.270	0.570
Benzo[*g*,*h*,*i*]perylene	126	0.001	35.000	1.380	0.375	0.252
Chrysene	127	0.036	43.000	1.987	0.528	0.372
Coronene	124	0.001	7.200	0.359	0.076	0.095
Cyclopenta[*c*,*d*]pyrene	122	0.003	0.620	0.062	0.008	0.034
Dibenz[*a*,*h*]anthracene	128	0.003	9.000	0.410	0.103	0.082
Fluoranthene	124	0.047	90.000	4.058	1.194	0.588
Fluorene	123	0.004	3.000	0.196	0.045	0.054
Indeno[1,2,3-*c*,*d*]pyrene	126	0.002	41.000	1.593	0.445	0.255
Naphthalene	114	0.001	42.000	1.175	0.498	0.068
Phenanthrene	124	0.038	43.000	2.343	0.633	0.416
Pyrene	124	0.042	69.000	3.111	0.907	0.490
Total PAHs[b]	112	0.404	554.03	28.335	8.072	4.477
Total LMW[c]	112	0.067	65.94	4.377	1.106	0.768
Total HMW[d]	121	0.335	505.06	22.835	6.587	3.796
Total B2 carcinogens[e]	126	0.141	268.000	11.673	3.358	1.902

[a] Standard error of the arithmetic mean.
[b] Total PAHs = the sum of the 18 PAHs. The number of observations included in total PAHs refers to only those where all of the 18 PAHs were measured.
[c] LMW, low molecular weight PAHs having two to three rings [116].
[d] HMW, high molecular weight PAHs having four or more rings [116].
[e] PAHs classified as probable human carcinogens by the US EPA [89].

pressure values (i.e., 10^{-4} to 10^{-3} range) and higher K_{ow} values (i.e., 10^5–10^6 range). Pyrene and fluoranthene have been suggested as potential source markers for incineration, wood burning and oil combustion [81], with the ratio of fluoranthene to pyrene providing information on PAH source. If the fluoranthene/pyrene index is greater than 1, the PAHs are considered to have been generated by pyrolytic processes, whereas if the index is less than 1, they are considered to be petrogenic in origin [73]. The mean ratio of fluoranthene to pyrene in the collected data is 1.25 ± 0.015 and 93% of the observations have a ratio greater than 1. Therefore, the PAHs detected in the SHD samples appear to be predominantly pyrolytic in origin.

The large range in PAH content is likely related to numerous factors that are thought to influence the levels of PAHs in SHD (see Table 5 and Section 2). Household characteristics including cigarette smoking, site location, type of flooring, and socio-economic status were examined to determine their influence on the PAH content of SHD. A paucity of information on socio-economic status and flooring type, prohibited statistical investigations of relationships between PAH content and these variables.

Two-way analysis of variance revealed significant effects ($r^2 = 0.15$–0.21, $p < 0.01$) of both smoking status and home location on PAH content (i.e., total PAH, LMW, HMW, or total B2 carcinogens), but failed to reveal a significant interaction between the home location and smoking status effects ($p > 0.15$). A subsequent one-way ANOVA using all the data confirmed a significant relationship ($0.02 > p < 0.04$) between the PAH content and the presence of smokers. However, separate analyses of the rural and urban data revealed that the relationship is only significant ($0.02 > p < 0.03$) for urban observations (Fig. 1, Table 7). One-way ANOVA also confirmed a

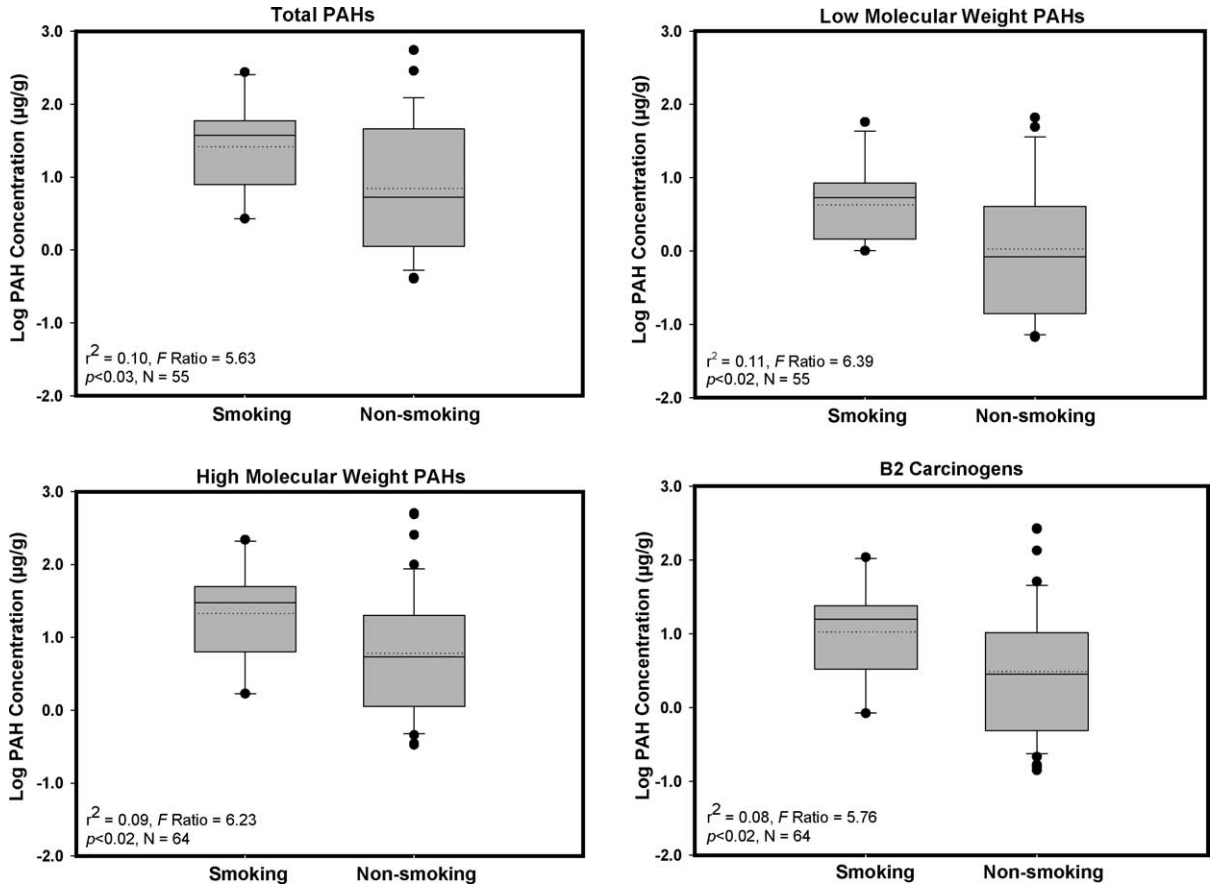

Fig. 1. Box plots showing the effect of cigarette smoking in urban locations on the contamination of SHD with total PAHs, low molecular weight PAHs, high molecular weight PAHs, and total B2 carcinogens.

significant relationship ($0.0002 > p < 0.02$) between the PAH content and house location (i.e., urban or rural) (Fig. 2, Table 7). Details of the ANOVA results for both effects are summarized in Table 7.

The smoking effect (Fig. 1) indicates that the PAH content of SHD from urban houses with smokers is 3.4–4 times higher than SHD samples from urban houses without smokers. The home location effect (Fig. 2) shows that the PAH content from houses in urban areas is 3–5 times higher than that collected in rural areas. Subsequent analyses of the deleted studentized residuals from each ANOVA revealed several significant outliers ($p < 0.05$). These outliers, all of which had positive residuals, were observations from urban homes in Columbus, OH sampled in 1992 and 1993 [39]. The total PAH composition of SHD samples from these homes (287.3–554.0 μg/g) is far higher than the geometric mean total PAH concentration (4.5 μg/g), and one of the sites (H08) yielded the highest total PAH value in the dataset. The authors of this study commented on the extremely high PAH content of SHD from this home, and noted that the home is located only one-quarter of a mile from a freeway, and road construction was underway during one of the sample collection periods.

The low r^2 values shown in Table 7 (8–11% for the smoking effect and 8–16% for the location effect) indicate that these effects only account for a small proportion of the total variation in the PAH content of SHD. The slightly higher r^2 and least-square means associated with the location effect suggest that location may be more important than smoking in

Table 7
ANOVA results summarizing the effects of house location and cigarette smoking on the PAH content of settled house dust

PAHs considered	Effect	r^2	F ratio	p
Total PAHs[a]	Smoking–urban only (smokers present/not present)	0.096	5.63	<0.03
Low molecular weight PAHs[b]		0.108	6.39	<0.02
High molecular weight PAHs[c]		0.090	6.23	<0.02
B2 carcinogenic PAHs[d]		0.085	5.76	<0.02
Total PAHs[a]	Location (urban/rural)	0.132	11.06	<0.002
Low molecular weight PAHs[b]		0.080	6.34	<0.02
High molecular weight PAHs[c]		0.149	14.34	<0.0003
B2 carcinogenic PAHs[d]		0.156	15.14	<0.0002

[a] Eighteen targeted PAHs (Table 5).
[b] PAHs having two to three rings [116].
[c] PAHs having four or more rings [116].
[d] PAHs classified as probable human carcinogens by the US EPA [89].

determining the PAH content of SHD. Investigations of PAHs in SHD have also noted differences between rural and urban (inner city) houses, with SHD from urban homes having markedly higher PAH concentrations [29]. In a pilot study of eight homes in Columbus, OH, Chuang et al. [28] noted that although the PAH content of SHD tended to be higher in the smokers' homes, smoking did not appear to be a primary determinant of PAH content in dust.

The weak effects of both smoking and location suggest that the PAH content of SHD is largely accounted for by other factors. Our inability to account for the effects of flooring type, and socio-economic status has already been noted. Some of the additional variability in PAH content is almost certainly attributable to a lack of a standardized dust collection protocol. For example, studies that employed the HVS3 sample collection method will likely contain the finer particles between 5 and 10 μm [37] and this will certainly affect the measured PAH content of SHD [31]. Moreover, variations in the deposition time between the last cleaning and the sample collection time can introduce variations in the chemical and physical properties of SHD. For a given deposition rate (mg/m^2 per day), the size of the collected SHD sample will depend on the interval between the last cleaning and the sampling time. This should not adversely affect PAH content measurements unless the concentrations drop below the analytical detection limit. However, the time interval between last cleaning and sampling could affect PAH determinations if contamination of the settling dust particles is temporally variable. Although several studies have shown that the concentration of suspended particulate matter in indoor air, and the deposition rate of house dust, is temporally variable and dependant on the magnitude, frequency and nature of household activities (e.g., cooking, cleaning, movement) [44,50,51], no studies have rigorously investigated temporal variability in the chemical content of SHD. Thus, for analyses of toxic substances, including mutagens and carcinogens, in SHD it is important to select a deposition period that provides sufficient sample for analysis, and integrates the temporal variability in deposition rate and contamination. The method recently published by the German Society of Engineers [55] recommends collection of 7-day time-integrated samples of settled dust (i.e., 7-day interval between thorough cleaning and sample collection).

3. The contribution of PAHs to the mutagenicity of settled house dust

PAH concentration data and published mutagenic potency values for each PAH were employed to calculate the predicted contribution of measured PAHs to the *Salmonella* mutagenic potency of SHD. The calculation assumed that the final mutagenic hazard of a given SHD sample containing a PAH mixture is the sum of that expected from each of the identified PAHs (i.e., the effects are additive). This assumption is supported by several published studies [82–85]. For example, White [85] showed that when PAH

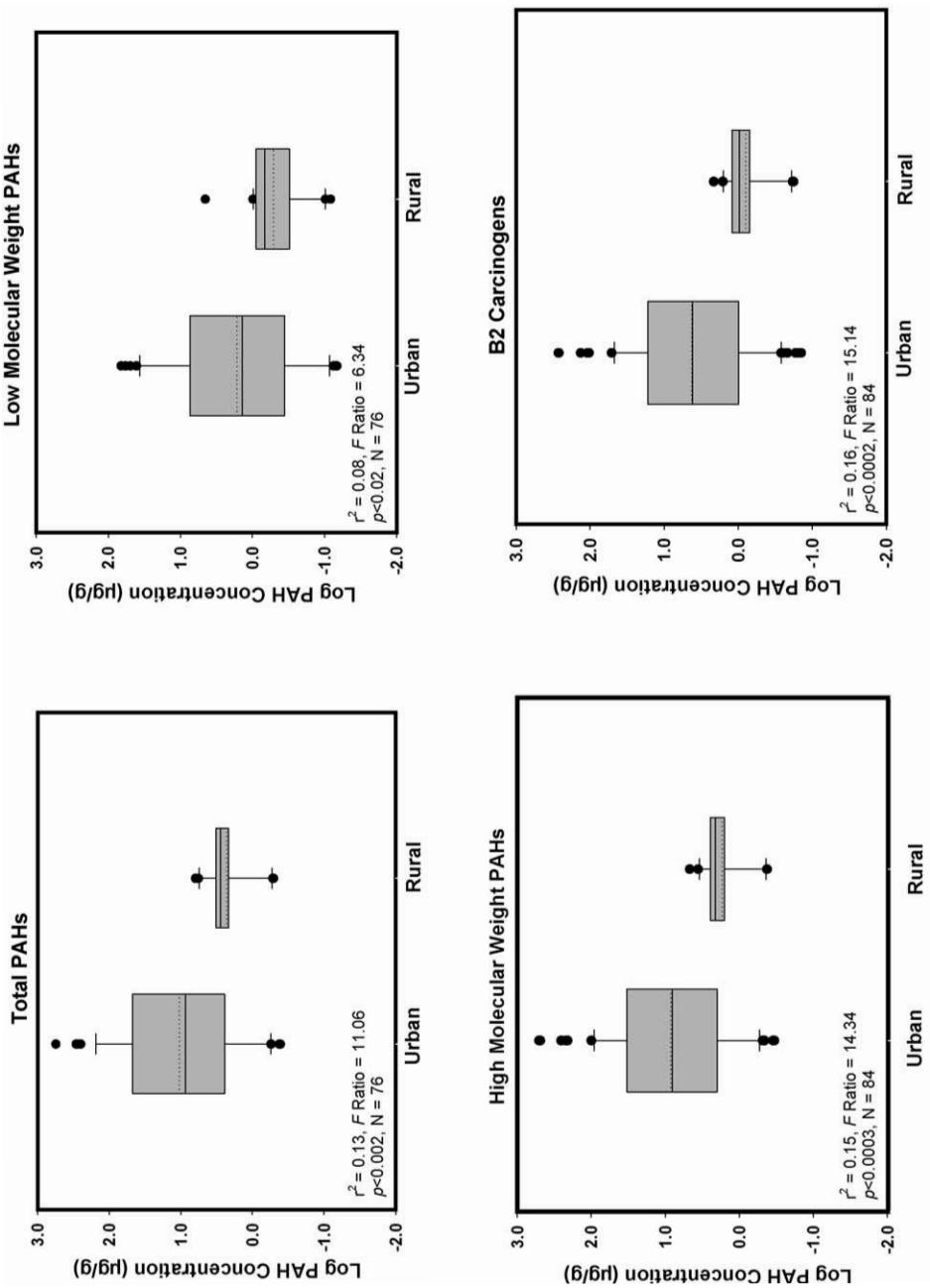

Fig. 2. Box plots showing the effect of house location (urban vs. rural) on the contamination of SHD with total PAHs, low molecular weight PAHs, high molecular weight PAHs, and total B2 carcinogens.

concentrations are low (<1 μg/assay ml) the total genotoxicity of PAH mixtures is largely determined by the sum of the effects from each of the mixture components (i.e., they are additive). Of the 18 PAHs targeted in the published dust studies, 14 are known to exhibit *Salmonella* mutagenic activity. The *Salmonella* mutagenic potency for each PAH and the predicted mutagenic potency for SHD extracts are summarized in Table 8.

The results obtained reveal that the commonly measured PAHs listed in Table 8 can be expected to contribute approximately 230 revertants/g of dust to *Salmonella* TA98 mutagenicity, and 301 revertants/g of dust to *Salmonella* TA100 mutagenicity. These potency values, attributable only to the listed PAHs, can be compared to the study by Roberts et al. [41] and the unpublished data for Ottawa homes. The predicted potency values (<302 revertants/g) are far lower than the measured potency values for actual dust extracts (1000–7000 TA98 revertants/g). Despite a paucity of mutagenicity measurements on actual samples of SHD, these results indicate that the measured PAHs are unable to account for more than 3–23% of the mutagenic hazard. Nevertheless, the presence of PAHs in SHD, and their carcinogenicity, is certainly cause for concern.

Table 8
Predicted *Salmonella* mutagenic potency of settled house dust based on the potency and mean concentration of selected PAHs

PAH	Mutagenic potency[a] + S9 (revertants/μg)		Dust PAH concentration (μg/g)[h]	Expected mutagenic potency (revertants/g)	
	TA98	TA100		TA98	TA100
Acenaphthene	NM	NM	0.032	–	–
Acenaphthylene	NA	NA	0.026	–	–
Anthracene	NM	0.243[b]	0.065	–	0.016
Benz[*a*]anthracene	56[c]	51.2	0.241	13.5	12.4
Benzo[*a*]pyrene	488	396	0.285	139	113
Benzo[*e*]pyrene	14.2	12.9	0.286	4.05	3.69
Benzo[*b*,*k*]fluoranthene[i]	60[c]	145[d]	0.570	34.2	82.4
Benzo[*g*,*h*,*i*]perylene	7.52	6.52	0.252	1.90	1.64
Chrysene	0.516	81.6	0.372	0.192	30.3
Coronene	33.1[e]	3.00[e]	0.095	3.15	0.286
Cyclopenta[*c*,*d*]pyrene	470[f]	523[f]	0.034	16.0	17.6
Dibenz[*a*,*h*]anthracene	39.0[c]	43.6	0.082	3.18	3.56
Fluoranthene	17.6	13.8	0.588	10.3	8.11
Fluorene	NM	NM	0.054	–	–
Indeno[1,2,3-*c*,*d*]pyrene	NA	79.1[g]	0.255	–	20.2
Naphthalene	NM	NM	0.068	–	–
Phenanthrene	1.38	1.20	0.416	0.576	0.499
Pyrene	8.32[e]	14.3[e]	0.490	4.07	7.01
Total				230	301

NM, not mutagenic in *Salmonella* mutagenicity assay; NA, data not available.

[a] Defined as the initial slope of the dose–response curve. Potency data taken from White and Rasmussen [117] except where indicated. All values were taken directly from the publications except for those marked [e] and [g] which were calculated by plotting the concentration response data and conducting linear regression analysis of the linear portion of the dose–response curve.

[b] Mortelmans et al. [111].

[c] Nagai et al. [118].

[d] Kubo et al. [119].

[e] Sakai et al. [120].

[f] Eisenstadt and Gold [121].

[g] Rice et al. [122].

[h] Geometric means of PAH data from 18 studies (see Table 6 and Appendix A).

[i] The potency value for benzo[*b*,*k*]fluoranthene was derived from the mean of the individual potency values for benzo[*b*]fluoranthene and benzo[*k*]fluoranthene.

4. Exposure to PAHs in settled house dust

Only a small number of studies have assessed human exposure to PAHs in SHD [23,24,29]. These studies have examined exposures to PAHs in dust in comparison to exposures via other media such as air or food. These assessments show that dietary ingestion of PAHs in food is often the primary exposure pathway for children. This holds true when considering exposure to both the sum of all targeted PAHs, or only those PAHs considered to be carcinogenic. When considering only the carcinogenic PAHs, non-dietary ingestion of PAHs in dust and soil is the second most important exposure route for children, and more important than inhalation.

Assessment of PAH exposure in children due to non-dietary ingestion of SHD indicates that toddlers playing on the floor and exhibiting hand-to-mouth behaviour can ingest more than 2.5 times as much as adults [18]. Since a child's body weight is only about one-fifth that of an average adult, childrens' exposures to PAHs in dust, in mg per kg body weight, are far greater than those recorded for adults. It is generally acknowledged that the higher surface area to body mass ratio, lower body weight, and relatively high intake of food, water and air (per unit body weight) contribute to higher exposures of children to certain contaminants. Therefore, the adverse health risks for children exposed to PAHs, especially via non-dietary ingestion, are generally believed to be considerably greater than for adults. Moreover, the early developmental stage of organ, immune and nervous systems in children are thought to contribute to an enhanced contaminant sensitivity [86].

At present, there are no reference doses or tolerable daily intake values for PAHs in SHD. The German Federal Environmental Agency's Commission for Indoor Air Quality has established the only guideline that currently exists. It states that exposure to levels above 10 μg of benzo[*a*]pyrene per gram of household dust should be minimized in order to prevent unspecified adverse health effects [87].

5. Cancer risk assessment of PAHs in settled house dust

The previously compiled PAH concentration data was used to assess excess lifetime cancer risk from non-dietary exposure of preschool aged children to carcinogenic PAHs in SHD.

The following equations were used to calculate excess lifetime cancer risk. Eq. (1) assesses the average lifetime daily dose.

$$\text{Lifetime average daily exposure dose} = \frac{C \times IR \times EF}{BW} \quad (1)$$

where C is the concentration of carcinogenic PAHs in the dust, in mg/g. The exposure calculations used the 5th percentile, geometric mean, median, and 95th percentile of the collected dust concentration values (see Table 9). IR is the ingestion rate: the amount of dust consumed via non-dietary ingestion in g/day. The value used was 0.1 g/day [56]. EF is the exposure factor: the fraction of an average person's lifetime that is occupied by the exposure period. These analyses

Table 9
PAH concentrations and PAH potency equivalency factors (PEF) used to calculate the excess lifetime cancer risks associated with non-dietary ingestion of carcinogenic PAHs in house dust by preschool aged children

Carcinogenic PAH	PAH concentration in dust (μg/g)				PEF[a]
	5th percentile	Geometric mean	Median	95th percentile	
Benzo[*a*]pyrene	0.023	0.285	0.195	13.000	1
Benz[*a*]anthracene	0.026	0.241	0.172	5.100	0.1
Benzo[*b*,*k*]fluroanthene	0.046	0.570	0.402	15.000	0.1
Chrysene	0.046	0.372	0.270	7.200	0.01
Indeno[1,2,3-*c*,*d*]pyrene	0.032	0.255	0.162	6.900	0.1
Dibenz[*a*,*h*]anthracene	0.012	0.082	0.058	1.800	5
Total carcinogenic PAHs	0.244	1.902	1.302	44.000	

[a] Potency equivalency factors taken from Collins et al. [123] except for dibenz[*a*,*h*]anthracene, which was obtained from Nisbet and LaGoy [124].

investigated a range of exposure factors representing various weekly exposure periods up to the age of 5 (i.e., preschool years between birth and 5th birthday). BW is the average body weight, in kg. A standard value of 13 kg was used [88].

Eq. (2) uses the average lifetime daily dose, the cancer slope factor and potency equivalency factors to calculate the excess lifetime cancer risk from exposure to carcinogenic PAHs in SHD.

$$\begin{aligned}\text{Lifetime cancer risk} = {} & \text{lifetime average daily exposure dose (mg/kg/day)} \\ & \times \text{slope factor (mg/kg/day)}^{-1} \\ & \times \text{potency equivalency factor} \end{aligned} \quad (2)$$

where the slope factor is the estimate of the probability of a response occurring per unit intake of the PAH over a lifetime. For these analyses, an oral slope factor for benzo[*a*]pyrene of 7.3 was used [89]. The potency equivalency factors are conversion factors used to express the potency of various PAHs in terms of benzo[*a*]pyrene equivalents (see Table 9).

With the exception of chrysene, only PAHs classified as IARC 2A or 2B carcinogens were included in the analyses. Although chrysene is classified as a class 3 carcinogen by IARC (limited evidence of carcinogenicity), it has been classified by the US EPA as a probable carcinogen (i.e., B2) and was therefore included. Naphthalene was excluded from the risk assessment calculations due to the lack of consensus by an NTP working group, and the designation of *not classifiable* by the US EPA, with respect to its carcinogenic hazard [89,90] (see Table 3). Table 10 contains a summary of the assumptions for the risk assessment calculations.

Table 10
Assumptions for assessment of excess cancer risk due to non-dietary exposure to PAHs in settled house dust by preschool aged children

Parameter	Assumptions and limitations
Dust contamination	The observed PAH concentrations in SHD are representative of the dust to which children are routinely exposed The PAH concentration is constant across time and space
Exposure	Exposures occur only before the age of 5 Exposures occur only in the home Ingestion of dust occurs only during waking hours The daily ingestion rate is constant across time, space and subject The collected dust samples are representative of the ingested material
Bioavailability	The PAHs in the collected dust samples are bioavailable and 100% is absorbed

Fig. 3 illustrates the relationship between the calculated excess lifetime cancer risk and the exposure rate (h/week) for several levels of PAH content derived from the collected data (i.e., 5th percentile, median, etc.). The results indicate that for the 5th percentile and median contamination levels even very high exposure rates do not yield excess cancer risk values that are appreciably above 1×10^{-6}. Only the extreme PAH levels denoted by the 95th percentile yield excess cancer risks appreciably above 1×10^{-6} for any exposure rate. At an exposure rate of 50 h/week, the calculated excess cancer risk is approximately

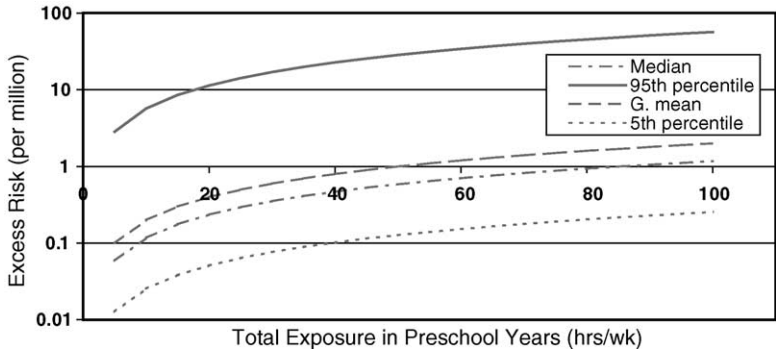

Fig. 3. Excess cancer risk due to non-dietary ingestion of PAHs in house dust during preschool years. Correction for enhanced risk due to childhood exposures was not applied.

28×10^{-6}. This value rises to more than 50×10^{-6} at exposure rates greater than 88 h/week. However, exposures of 88 h/week may not be likely since that would equate to more than 12 waking hours per day of SHD exposure. Closer inspection of the data confirmed that the highest risk values correspond to exposures to the highly contaminated SHD samples (i.e., total PAH > 250 μg/g) collected from homes located in highly urban areas in Columbus, OH (see Section 2.3) [39].

It should be noted that the risk estimates did not attempt to account for enhanced risk that may be associated with exposures that occur early in life (i.e., younger than 15 years of age). Factors such as incomplete immune system development, rapid growth, enhanced rates of cell division, and hormonal fluctuations are thought to contribute to an enhanced lifetime risk of cancer from early life stage (i.e., childhood) exposures [91,92]. In a recent comprehensive guidance document currently being considered by a review panel of the EPA Science Advisory Board [93], the US EPA has proposed that, for exposures to carcinogens with mutagenic modes of action, risk estimates should be adjusted by a factor of 10 for exposures that occur before the age of 2, and a factor of 3 for exposures that occur between the ages of 2 and 15. In the context of the risk assessment conducted here, the safety factors being considered in the US EPA guidance document would translate into a 10-fold increase in excess lifetime risk for 40% of the 5 year exposure period (i.e., birth to second birthday), and a 3-fold increase in risk for 60% of the exposure period (i.e., second to fifth birthday). This would result in a composite weighting factor for the preschool years of 5.2. If this adjustment is applied to the aforementioned risk estimates, the final values associated with exposures to 95th percentile PAH levels correspond to risk estimates of 146×10^{-6} for 50 h per week (∼7 h/day) and 260×10^{-6} for 88 h per week (∼12.5 h/day). Based on US EPA current risk assessment guidelines [94], these values would be approximately 1.5–2.5-fold times the range of acceptable excess lifetime cancer risk (i.e., 10^{-6} to 10^{-4}).

Thus, the risk assessment calculations indicate that, with the exception of circumstances whereby PAH concentrations are exceptionally high and adjustments are made for early life exposure, regulatory agencies such as the US EPA would deem the excess lifetime cancer risk from preschool, non-dietary ingestion of carcinogenic PAHs in SHD as acceptable.

Numerous investigators have suggested a number of simple precautionary measures to reduce exposures to PAHs and other contaminants in SHD, and consequently reduce the risk of adverse effects. These measures include use of a door mat to reduce the quantity of particle-bound pollutants tracked in from outside, removal of shoes inside the home, appropriate ventilation, installation of air-filters, as well as frequent and appropriate cleaning of carpets and floors [95].

6. Conclusions

House dust is a complex mixture of particulate materials of both natural and anthropogenic origin. Although it is commonly seen as a simple nuisance, it can also play an important role in the exposure of humans to toxic contaminants. Dust can act as a reservoir for semi-volatile organic compounds such as polycyclic aromatic hydrocarbons that adsorb to suspended particulates and are deposited on indoor surfaces (e.g., furniture, floors). The chemical composition, physical structure, and potential hazard of settled house dust is dependant on a wide range of factors that govern the penetration of particulate material from outdoor environments (e.g., soil and suspended particulates), the magnitude and nature of indoor activities that can generate toxic substances (e.g., combustion, cooking), the magnitude and nature of indoor activities (e.g., movement and cleaning), and the magnitude and frequency of dust exposure (e.g., ingestion, inhalation).

Studies available to date indicate that SHD contains a variety of substances that are known to be mutagenic (e.g., PAHs and related compounds). Despite the presence of these mutagenic PAHs, it appears that less than 25% of the mutagenic activity measured on the *Salmonella* mutagenicity assay can be accounted for by the mutagenic PAHs selected for chemical analysis. However, it is readily apparent that there is a paucity of information on the sources, hazards and fate of potential mutagens in SHD. Research currently underway at Health Canada in Ottawa is addressing these deficiencies, and preliminary results indicate that some of the direct acting (*Salmonella*) mutagenic

activity may be accounted for by nitro-arenes, compounds that are often associated with diesel emissions. The portion of the S9-activated mutagenicity that cannot be accounted for by the frequently measured PAHs, are potentially attributable to the presence of non-target PAHs, heterocyclic compounds, and PAH derivatives that are not measured during routine chemical analyses.

Additional analyses of data showed that an urban location, and the presence of cigarette smokers, increases the PAH content of SHD. However, the detected empirical effects of home location and cigarette smoking are weak, and the identification of other factors (e.g., flooring type, season, deposition rate, ventilation, socio-economic status) that may affect the PAH content of SHD seems a promising area for further research. Moreover, further research investigating the temporal variability of dust contamination, the sources and fate of PAHs in SHD, and the size spectrum of resuspended particles, would improve the ability to collect representative samples.

The estimated excess lifetime cancer risks from non-dietary ingestion of PAHs in settled house dust by preschool aged children appears to be predominantly in the range that is acknowledged as acceptable by regulatory agencies such as the US EPA (i.e., 10^{-6} to 10^{-4}). Substantially elevated risk estimates in the range 1.5×10^{-4} to 2.5×10^{-4} correspond only to situations where the PAH content is at or beyond the 95th percentile, and the risk estimates are adjusted for enhanced susceptibility at early life stages. More detailed investigations of high PAH SHD samples from inner city homes; including source apportionment of carcinogenic PAHs, is an area that requires further attention. Moreover, additional research should continue to address issues of early life stage susceptibility and rates of childhood exposure to carcinogens in SHD. More precise assessments of childhood exposure rates via non-dietary ingestion, inhalation and dermal adsorption, as well as information on adsorption rates, would permit more accurate and thorough characterizations of the carcinogenic hazards posed by PAHs and other substances in SHD.

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Appendix A

PAH concentrations (ug/g) in settled house dust samples from 18 published studies

Reference	Sampling area[a]	Site	Collection method	Location	Smoking	Income	ACNP[b]	ACNPHT	ANTH	BAA	BAP	BEP	BBKF	BGHIP	CHRY	CORO	CPCDP	DBAHA	FLUORAN	FLUOR	I123CDP	NAPH	PHEN	PYR
[22]	ME	Mean	Vacuum	Unknown	Unknown	Unknown			0.107	0.954	1.58													3.12
[23]	NC	D03_1	HVS3	Rural	No	Low	0.084	0.01	0.092	0.345	0.403	0.319	0.423	0.368	0.424	0.104	0.11	0.144	0.908	0.042	0.396	0.046	0.703	0.721
	NC	D03_2	HVS3	Rural	No	Low	0.068	0.011	0.071	0.264	0.291	0.243	0.317	0.29	0.324	0.088	0.085	0.111	0.699	0.031	0.303	0.041	0.533	0.57
	NC	DO9_1	HVS3	Urban	No	Unknown	0.011	0.004	0.011	0.031	0.045	0.048	0.054	0.059	0.056	0.025	0.013	0.023	0.093	0.016	0.055	0.013	0.078	0.081
	NC	DO9_2	HVS3	Urban	No	Unknown	0.005	0.003	0.005	0.022	0.023	0.027	0.03	0.036	0.046	0.017	0.01	0.015	0.049	0.01	0.032	0.008	0.038	0.042
	NC	HA3	HVS3	Rural	No	Low	0.005	0.004	0.011	0.019	0.026	0.034	0.037	0.041	0.05	0.02	0.01	0.016	0.074	0.007	0.035	0.006	0.064	0.057
	NC	HB3	HVS3	Rural	No	Low	0.004	0.002	0.008	0.025	0.024	0.031	0.036	0.04	0.037	0.022	0.013	0.02	0.076	0.006	0.036	0.006	0.056	0.062
	NC	HC3	HVS3	Rural	Yes	Low	0.01	0.003	0.015	0.059	0.07	0.075	0.096	0.075	0.086	0.026	0.022	0.032	0.185	0.009	0.076	0.007	0.108	0.142
	NC	HD3	HVS3	Rural	No	Low	0.008	0.003	0.012	0.033	0.042	0.045	0.055	0.058	0.061	0.023	0.014	0.027	0.093	0.009	0.053	0.006	0.065	0.073
	NC	HE9	HVS3	Urban	No	Unknown	0.004	0.003	0.006	0.023	0.037	0.038	0.046	0.051	0.041	0.026	0.01	0.02	0.077	0.005	0.048	0.005	0.044	0.061
	NC	HF9	HVS3	Urban	No	Unknown	0.019	0.023	0.066	0.519	0.768	0.809	0.967	0.961	0.838	0.32	0.172	0.294	1.555	0.028	0.963	0.035	0.596	1.202
	NC	HG9	HVS3	Urban	No	Unknown	0.007	0.006	0.013	0.054	0.086	0.092	0.108	0.116	0.225	0.065	0.026	0.044	0.239	0.012	0.108	0.001	0.143	0.181
	NC	HH9	HVS3	Urban	No	Unknown	0.008	0.007	0.012	0.042	0.084	0.095	0.11	0.112	0.09	0.047	0.024	0.044	0.203	0.01	0.112	0.006	0.128	0.155
	NC	HI9	HVS3	Urban	No	Unknown	0.005	0.005	0.011	0.04	0.07	0.076	0.089	0.101	0.097	0.056	0.022	0.041	0.168	0.01	0.094	0.015	0.093	0.129
[25]	MI, IA, LA, WA	Median	Vacuum	Unknown	Unknown	Unknown				0.136	0.154		0.409		0.27			0.036			0.161			
[26]	NY	Median	HVS3	Unknown	Unknown	Unknown				1.14	1.46							0.29						
[32]	MA	Mean	Vacuum	Unknown	Unknown	Unknown				2.91	2.9													
[24]	NC	D01	HVS3	Rural	No	Low	0.022	0.003	0.038	0.155	0.208	0.128	0.15	0.099	0.206	0.011	0.045	0.031	0.305	0.019	0.095	0.015	0.875	0.258
	NC	D02	HVS3	Urban	No	Low	0.02	0.018	0.076	0.491	0.688	0.627	0.762	0.497	1.213	0.136	0.145	0.109	1.827	0.019	0.48	0.015	0.637	1.457
	NC	D03	HVS3	Rural	No	Low	0.05	0.005	0.053	0.258	0.203	0.132	0.159	0.112	0.318	0.015	0.072	0.034	0.536	0.019	0.119	0.026	0.343	0.45
	NC	D04	HVS3	Urban	No	Unknown	0.006	0.006	0.014	0.052	0.143	0.064	0.078	0.04	0.102	0.004	0.018	0.014	0.178	0.006	0.04	0.006	0.092	0.14
	NC	D05	HVS3	Urban	No	Unknown	0.009	0.002	0.007	0.031	0.117	0.046	0.037	0.003	0.046	0.001	0.014	0.01	0.082	0.006	0.003	0.009	0.06	0.066
	NC	D06	HVS3	Urban	No	Low	0.009	0.002	0.008	0.036	0.098	0.042	0.042	0.015	0.066	0.001	0.016	0.008	0.077	0.004	0.013	0.003	0.049	0.069
	NC	D07	HVS3	Urban	No	Unknown	0.003	0.001	0.009	0.031	0.034	0.025	0.032	0.006	0.036	0.001	0.011	0.003	0.084	0.004	0.005	0.001	0.051	0.067
	NC	D08	HVS3	Urban	No	Unknown	0.011	0.005	0.013	0.075	0.131	0.085	0.1	0.043	0.132	0.002	0.03	0.012	0.18	0.007	0.035	0.005	0.098	0.149
	NC	D09	HVS3	Urban	No	Unknown	0.006	0.001	0.007	0.038	0.015	0.015	0.061	0.001	0.039	0.001	0.016	0.003	0.085	0.007	0.002	0.002	0.05	0.066
	NC	D10	HVS3	Urban	No	Unknown	0.001	0.01	0.019	0.562	0.82	0.662	0.861	0.725	1.19	0.2	0.146	0.207	1.141	0.009	0.786	0.008	0.312	0.898
[29]	NC	pilot1	HVS3	Urban	No	Low	0.139	0.167	0.277	0.694	0.586	0.746	1.341	0.607	2.406	0.065	0.220	0.257	1.889	1.220	0.699	0.090	2.149	1.646
	NC	pilot2	HVS3	Urban	Yes	Low	0.011	0.059	0.101	0.234	0.169	0.351	0.590	0.300	0.770	0.055	0.119	0.108	0.714	0.080	0.299	0.044	0.839	0.738
	NC	wrns1	HVS3	Rural	No	Low	0.049	0.104	0.042	0.126	0.139	0.143	0.346	0.197	0.217	0.204	0.061	0.086	0.445	0.051	0.172	0.085	0.284	0.337
	NC	wrns2	HVS3	Rural	No	Low	0.040	0.043	0.167	0.099	0.070	0.149	0.237	0.169	0.141	0.501	0.060	0.072	0.245	0.106	0.103	0.293	0.288	0.236
	NC	wrns3	HVS3	Rural	No	Low	0.012	0.008	0.021	0.103	0.104	0.115	0.343	0.147	0.139	0.073	0.049	0.060	0.333	0.053	0.110	0.056	0.142	0.205
	NC	wrns4	HVS3	Rural	No	Low	0.009	0.017	0.015	0.061	0.112	0.569	0.325	0.108	0.100	0.037	0.027	0.022	0.155	0.028	0.072	4.299	0.133	0.115
	NC	wuns1	HVS3	Urban	No	Low	0.177	0.090	0.027	0.337	0.286	0.361	0.745	0.240	0.360	0.062	0.126	0.088	0.764	0.144	0.218	0.315	0.425	0.754
	NC	wuns2	HVS3	Urban	No	Low	0.004	0.023	0.070	0.039	0.086	0.050	0.171	0.120	0.046	0.064	0.060	0.068	0.092	0.024	0.047	0.017	0.206	0.059
	NC	wuns3	HVS3	Urban	No	Low	0.040	0.075	0.083	0.358	0.254	0.252	0.515	0.224	0.293	0.062	0.079	0.089	0.442	0.075	0.224	0.076	0.416	0.335
	NC	wuns4	HVS3	Urban	No	Low	0.026	0.027	0.039	0.152	0.151	0.290	1.065	0.560	0.308	0.219	0.125	0.213	0.478	0.157	0.562	0.041	0.582	0.314
	NC	wuns5	HVS3	Urban	No	Low	0.146	0.089	0.032	0.340	0.285	0.381	0.763	0.245	0.301	0.086	0.106	0.115	0.682	0.135	0.235	0.315	0.376	0.612
	NC	suns1	HVS3	Urban	No	Low	0.043	0.272	0.161	0.602	0.633	0.420	1.052	0.583	0.780	0.260	0.209	0.414	0.969	0.105	0.620	0.316	0.483	0.725
	NC	suns2	HVS3	Urban	No	Low	0.038	0.168	0.046	0.095	0.069	0.108	0.243	0.091	0.187	0.097	0.038	0.039	0.300	0.049	0.084	0.155	0.342	0.303
	NC	suns3	HVS3	Urban	No	Low	0.039	0.074	0.096	0.167	0.289	0.221	0.516	0.286	0.355	0.151	0.072	0.073	0.532	0.069	0.227	0.136	0.394	0.396
	NC	suns4	HVS3	Urban	No	Low	0.051	0.053	0.063	0.218	0.236	0.258	0.665	0.364	0.351	0.214	0.072	0.077	0.501	0.081	0.278	0.121	0.515	0.378
	NC	suns5	HVS3	Urban	No	Low	0.035	0.064	0.068	0.171	0.281	0.272	0.587	0.297	0.405	0.160	0.049	0.088	0.500	0.057	0.254	0.280	0.377	0.439
	NC	srns1	HVS3	Rural	No	Low	0.051	0.051	0.131	0.172	0.208	0.186	0.424	0.214	0.323	0.062	0.036	0.050	0.489	0.072	0.182	0.242	0.388	0.339
	NC	srns2	HVS3	Rural	No	Low	0.020	0.018	0.028	0.111	0.186	0.153	0.366	0.158	0.237	0.068	0.036	0.048	0.359	0.038	0.139	0.060	0.195	0.268
	NC	srns3	HVS3	Rural	No	Low	0.023	0.034	0.040	0.414	0.141	0.144	0.453	0.166	0.204	0.061	0.065	0.054	0.280	0.056	0.146	0.262	0.259	0.209
	NC	srns4	HVS3	Rural	No	Low	0.035	0.023	0.058	0.14	0.229	0.176	0.408	0.169	0.279	0.054	0.074	0.055	0.495	0.048	0.151	0.127	0.368	0.349
	NC	4suns1	HVS3	Urban	Yes	Low	0.055	0.033	0.748	0.452	0.483	0.429	1.118	0.477	0.644	0.231	0.145	0.143	0.967	0.063	0.496	0.211	0.44	0.756
	NC	4suns2	HVS3	Urban	Yes	Low	0.055	0.116	0.282	0.07	0.24	0.106	0.258	0.101	0.167	0.127	0.025	0.027	0.251	0.091	0.075	0.152	0.312	0.236
	NC	4srs	HVS3	Rural	Yes	Low	0.03	0.128	0.053	0.108	0.195	0.099	0.208	0.079	0.148	0.039	0.035	0.015	0.221	0.081	0.072	0.233	0.238	0.38
	NC	4srs2	HVS3	Rural	Yes	Low	0.034	0.071	0.124	0.112	0.162	0.144	0.384	0.139	0.272	0.056	0.027	0.037	0.378	0.057	0.126	0.085	0.307	0.292
[30]	Germany	Median 1	Vacuum, sweeping, wiping	Various	Various	Various					0.3													

Appendix A. (*Continued*)

Reference	Sampling area[a]	Site	Collection method	Location	Smoking	Income	ACNP[b]	ACNPHT	ANTH	BAA	BAP	BEP	BBKF	BGHIP	CHRY	CORO	CPCDP	DBAHA	FLUORAN	FLUOR	I123CDP	NAPH	PHEN	PYR
[31]	NC	Composite	Vacuum	Suburban	Unknown	Middle				1.82	1.76		5.52	2.73	3.41	1.42		0.48			2.33			
[38]	Germany	Median	Vacuum	Unknown	Unknown	Unknown																	0.3	
[35]	NC	F	HVS3	Urban	Yes	Low	0.055	0.033	0.75	0.45	0.48	0.43	1.1	0.48	0.64	0.23	0.14	0.14	0.97	0.063	0.5	0.21	0.44	0.76
	NC	G	HVS3	Urban	Yes	Low	0.055	0.12	0.28	0.07	0.24	0.11	0.26	0.1	0.17	0.13	0.025	0.027	0.25	0.091	0.075	0.15	0.31	0.24
	NC	K	HVS3	Rural	Yes	Low	0.03	0.13	0.053	0.11	0.2	0.099	0.21	0.079	0.15	0.039	0.035	0.015	0.22	0.081	0.072	0.23	0.24	0.38
	NC	M	HVS3	Rural	Yes	Low	0.034	0.071	0.12	0.11	0.16	0.14	0.38	0.14	0.27	0.056	0.027	0.037	0.38	0.057	0.13	0.085	0.31	0.29
[34]	NC	A-HD-X	No data	Unknown	Unknown	Low	0.202	0.131	0.116	0.36	0.138	0.207	0.473	0.122	0.367	0.031	0.1	0.032	1.025	0.814	0.117	0.212	0.741	0.769
	NC	B-HD-X	No data	Unknown	Unknown	Low	0.015	0.022	0.021	0.087	0.037	0.284	0.242	0.07	0.132	0.03	0.02	0.018	0.299	0.029	0.076	0.077	0.197	0.228
	NC	C-HD-X	No data	Unknown	Unknown	Low	0.019	0.016	0.025	0.041	0.022	0.066	0.116	0.035	0.049	0.016	0.008	0.012	0.14	0.033	0.025	0.032	0.143	0.095
	NC	D-HD-X	No data	Unknown	Unknown	Low	0.032	0.023	0.038	0.117	0.063	0.123	0.255	0.056	0.128	0.044	0.018	0.014	0.208	0.058	0.065	0.044	0.237	0.141
	NC	E-HD-X	No data	Unknown	Unknown	Low	0.022	0.03	0.025	0.144	0.073	0.128	0.239	0.101	0.162	0.065	0.036	0.017	0.2	0.033	0.106	0.082	0.202	0.157
	NC	F-HD-X	No data	Unknown	Unknown	Low	0.185	0.063	0.331	1.465	0.931	0.907	2.452	0.817	1.052	0.283	0.404	0.24	2.148	0.19	0.879	0.136	1.316	1.571
	NC	G-HD-X	No data	Unknown	Unknown	Low	0.028	0.042	0.025	0.117	0.04	0.137	0.146	0.069	0.166	0.032	0.025	0.014	0.237	0.049	0.052	0.039	0.173	0.184
	NC	H-HD-X	No data	Unknown	Unknown	Low	0.043	0.026	0.036	0.323	0.126	0.149	0.402	0.124	0.43	0.039	0.065	0.036	0.627	0.05	0.143	0.155	0.318	0.407
	NC	I-HD-X	No data	Unknown	Unknown	Low	0.007	0.007	0.007	0.064	0.03	0.04	0.105	0.046	0.067	0.035	0.013	0.016	0.129					
	NC	J-HD-X	No data	Unknown	Unknown	Low	0.019	0.015	0.019	0.09	0.062	0.101	0.215	0.083	0.106	0.038	0.026	0.02	0.249	0.029	0.088	0.061	0.137	0.169
	NC	K-HD-X	No data	Unknown	Unknown	Low	0.055	0.023	0.029	0.187	0.09	0.241	0.365	0.122	0.171	0.037	0.045	0.031	0.491	0.027	0.134	0.026	0.183	0.334
	NC	L-HD-X	No data	Unknown	Unknown	Low	0.009	0.01	0.036	0.209	0.293	0.043	0.765	0.322	0.225	0.12	0.061	0.076	0.498	0.035	0.338	0.018	0.237	0.347
	NC	M-HD-X	No data	Unknown	Unknown	Low	0.037	0.046	0.022	0.108	0.044	0.11	0.205	0.069	0.126	0.039	0.022	0.019	0.272	0.069	0.074	0.05	0.225	0.185
[34,96]	AZ	514125	No data	Unknown	Unknown	Unknown	0.024	0.013	0.024	0.04	0.042	0.06	0.101	0.058	0.124	0.034	0.01	0.013	0.127	0.029	0.058	0.053	0.099	0.102
	AZ	181831	No data	Unknown	Unknown	Unknown	0.015	0.007	0.017	0.111	0.143	0.148	0.357	0.148	0.146	0.054	0.024	0.036	0.282	0.02	0.164	0.041	0.17	0.239
	AZ	314275	No data	Unknown	Unknown	Unknown	0.027	0.031	0.04	0.253	0.421	0.457	1.13	0.484	0.47	0.187	0.071	0.117	0.68	0.026	0.532	0.041	0.244	0.569
	AZ	314985	No data	Unknown	Unknown	Unknown	0.017	0.014	0.021	0.054	0.067	0.107	0.254	0.105	0.183	0.05	0.012	0.029	0.202	0.025	0.113	0.044	0.156	0.164
	AZ	318479	No data	Unknown	Unknown	Unknown	0.015	0.014	0.019	0.056	0.069	0.103	0.228	0.083	0.129	0.034	0.011	0.021	0.148	0.019	0.088	0.039	0.084	0.138
	AZ	523527	No data	Unknown	Unknown	Unknown	0.017	0.013	0.048	0.033	0.063	0.11	0.211	0.091	0.121	0.056	0.008	0.026	0.143	0.025	0.104	0.025	0.074	0.123
	AZ	313546	No data	Unknown	Unknown	Unknown	0.027	0.041	0.022	0.044	0.059	0.089	0.179	0.102	0.093	0.096	0.011	0.026	0.13	0.028	0.102	0.035	0.099	0.114
	AZ	181815	No data	Unknown	Unknown	Unknown	0.014	0.001	0.024	0.072	0.072	0.126	0.295	0.098	0.101	0.062	0.013	0.018	0.225	0.026	0.106	0.032	0.125	0.188
	AZ	312413	No data	Unknown	Unknown	Unknown	0.005	0.012	0.05	0.033	0.06	0.13	0.199	0.094	0.075	0.087	0.011	0.023	0.11	0.025	0.1	0.047	0.063	0.091
	AZ	513278	No data	Unknown	Unknown	Unknown	0.017	0.013	0.038	0.468	0.68	0.578	1.578	0.636	0.685	0.215	0.148	0.158	0.746	0.019	0.727	0.02	0.237	0.72
	AZ	312572	No data	Unknown	Unknown	Unknown	0.023	0.008	0.012	0.032	0.023	0.051	0.075	0.032	0.074	0.071	0.007	0.027	0.1	0.009	0.032	0.058	0.216	0.092
	AZ	317968	No data	Unknown	Unknown	Unknown	0.014	0.007	0.014	0.022	0.018	0.126	0.145	0.047	0.074	0.025	0.003	0.012	0.102	0.018	0.049	0.029	0.082	0.081
	AZ	323619	No data	Unknown	Unknown	Unknown	0.013	0.023	0.022	0.044	0.063	0.088	0.2	0.101	0.101	0.074	0.012	0.024	0.162	0.049	0.102	0.064	0.19	0.146
	AZ	321583	No data	Unknown	Unknown	Unknown	0.039	0.023	0.028	0.106	0.112	0.193	0.666	0.199	0.186	0.16	0.023	0.05	0.281	0.054	0.222	0.095	0.172	0.231
	AZ	315148	No data	Unknown	Unknown	Unknown	0.032	0.018	0.021	0.11	0.131	0.12	0.358	0.173	0.195	0.109	0.027	0.055	0.313	0.04	0.205	0.09	0.218	0.277
	AZ	323895	No data	Unknown	Unknown	Unknown	0.009	0.015	0.13	0.039	0.035	0.054	0.113	0.061	0.078	0.055	0.004	0.036	0.081	0.02	0.066	0.082	0.078	0.085
	AZ	315845	No data	Unknown	Unknown	Unknown	0.011	0.007	0.015	0.037	0.051	0.031	0.093	0.045	0.053	0.032	0.018	0.013	0.072	0.01	0.049	0.017	0.041	0.06
	AZ	324319	No data	Unknown	Unknown	Unknown	0.017	0.01	0.026	0.057	0.067	0.081	0.252	0.106	0.096	0.065	0.015	0.039	0.139	0.021	0.12	0.045	0.073	0.12
	AZ	513656	No data	Unknown	Unknown	Unknown	0.013	0.008	0.014	0.026	0.02	0.123	0.127	0.033	0.082	0.015	0.006	0.013	0.098	0.017	0.043	0.037	0.063	0.075
	AZ	323521	No data	Unknown	Unknown	Unknown	0.009	0.005	0.005	0.017	0.021	0.062	0.126	0.043	0.055	0.035	0.005	0.009	0.047	0.013	0.05	0.022	0.04	0.043
	AZ	181525	No data	Unknown	Unknown	Unknown	0.009	0.004	0.008	0.142	0.093	0.049	0.09	0.04	0.048	0.021	0.003	0.01	0.079	0.011	0.045	0.006	0.071	0.063
	AZ	319179	No data	Unknown	Unknown	Unknown	0.011	0.005	0.009	0.211	0.04	0.069	0.138	0.072	0.101	0.04	0.008	0.016	0.101	0.012	0.078	0.01	0.044	0.086
[33]	TX	Mean1	HVS3	Various	Various	Low	0.0100		0.1820	0.1030	0.1280		0.2700	0.1300	0.1930			0.0321	0.2750	0.0216	0.1400	0.1880	0.1790	0.2420
	TX	Mean2	HVS3	Various	Various	Low	0.0182		0.0278	0.0824	0.1108		0.2000	0.1038	0.1455			0.0258	0.2375	0.0169	0.1090	0.3000	0.2495	0.1962
[36]	KY	HD31	HVS3	Unknown	Unknown	Unknown	0.011	0.055	0.582	0.223	0.189	0.227	0.522	0.228	0.356	0.13	0.073	0.098	0.325	0.069	0.21	0.093	0.25	0.27
	KY	HD32	HVS3	Unknown	Unknown	Unknown	0.011	0.041	0.042	0.18	0.177	0.2	0.492	0.186	0.276	0.061	0.064	0.061	0.324	0.035	0.184	0.027	0.2	0.249
	KY	HD33	HVS3	Unknown	Unknown	Unknown	0.043	0.191	0.051	0.145	0.131	0.114	0.321	0.151	0.155	0.085	0.051	0.06	0.293	0.061	0.149	0.065	0.287	0.212
[27]	IL, IN, IA, MI, MN, NJ, OH, PA, WI	Median	HVS3	Unknown	Unknown	Unknown				0.71	1.12		2.34	0.71	1.26	0.28		0.36			0.72			
[37]	WA	HD1	HVS3	Urban	No	Unknown			0.062	0.32	0.49	0.51	1	0.44	0.53	0.14	0.01	0.15	0.75	0.047	0.35		0.6	0.66
	WA	HD2	HVS3	Urban	No	Unknown			0.09	0.41	0.6	0.54	1.1	0.5	0.7	0.11	0.01	0.093	1.1	0.062	0.41		0.75	0.93
	WA	HD3	HVS3	Urban	No	Unknown			0.10	0.68	0.76	0.87	2.2	0.94	1.2	0.22	0.01	0.26	1.5	0.12	0.77		1.4	1.4
	WA	HD7	HVS3	Urban	No	Unknown			0.11	0.44	0.52	0.68	1.6	0.68	0.86	0.48	0.01	0.21	1.2	0.06	0.59		0.91	1.2
	WA	HD9	HVS3	Urban	No	Unknown			0.084	0.32	0.49	0.41	0.88	0.36	0.54	0.065	0.01	0.073	1	0.053	0.33		1	0.91
	WA	HD10	HVS3	Urban	No	Unknown			0.091	0.42	0.61	0.67	1.2	0.66	0.95	0.37	0.06	0.51	0.86	0.039	0.65		0.76	0.97
	WA	HD13	HVS3	Urban	No	Unknown			0.063	0.25	0.46	0.38	0.77	0.37	0.52	0.11	0.01	0.16	0.76	0.053	0.37		1	0.68
	WA	HD14	HVS3	Urban	No	Unknown			0.40	1.5	1.7	1.5	3.5	1.3	2.4	0.14	0.01	0.32	3.9	0.28	1.4		3.1	3

	WA	HD15	HVS3	Urban	No	Unknown		0.10	0.54	0.92	0.74	1.6	0.83	0.98	0.14	0.05	0.15	1.6	0.089	0.81		1.1	1.4
	OH	H01DS_Jun	HVS3	Urban	Yes	Unknown	0.28	0.2	0.73	3.3	13	3.6	10	4.6	5.7	2.2	0.21	1.8	12	0.48	5.5	0.01	6.8	9.4
[39]	OH	H01DS_Oct	HVS3	Urban	Yes	Unknown	0.24	0.21	0.65	3.3	3.7	3.1	7.8	3.1	5.1	0.81	0.07	0.92	10	0.48	3.5	3	6.2	7.9
	OH	H01DS_Apr	HVS3	Urban	Yes	Unknown	0.15	0.12	0.44	2	3	2.5	5.7	2.4	3	0.25	0.022	0.5	6.7	0.31	2.7	0.03	4.3	4.8
	OH	H02DN_Jun	HVS3	Urban	No	Unknown	0.09	0.12	0.25	1.3	21	1.7	3.7	2.4	2.2	1.4	0.17	0.96	3.7	0.15	2.7	0.16	2.1	2.9
	OH	H02DN_Oct	HVS3	Urban	No	Unknown	0.08	0.03	0.16	1.5	1.5	1.5	3.5	1.2	2.1	0.3	0.03	0.44	3.1	0.18	1.3	32	2.1	2.6
	OH	H02DN_Apr	HVS3	Urban	No	Unknown	0.13	0.036	0.29	1.6	2.1	1.9	4.4	1.6	2.5	0.11	0.01	0.28	5	0.23	1.8	0.068	3.3	3.7
	OH	H03DN_Jun	HVS3	Urban	No	Unknown	0.07	0.04	0.16	1	1.8	1.6	3.5	1.5	1.6	0.7	0.04	0.67	3.2	0.11	1.8	0.09	2.1	2.5
	OH	H03DN_Oct	HVS3	Urban	No	Unknown	0.11	0.07	0.28	1.8	2.4	2.1	5.8	2.5	2.7	0.52	0.02	0.81	6.1	0.2	2.6	9.2	3.7	4.8
	OH	H03DN_Apr	HVS3	Urban	No	Unknown	0.1	0.17	0.27	1.9	4.2	3.1	8.1	3.7	2.2	2.1	0.62	1.8	5.2	0.17	5.4	0.066	2.7	4.1
	OH	H04DS_Jun	HVS3	Urban	Yes	Unknown	0.05	0.04	0.11	0.48	0.7	11	1.6	1	1	0.5	0.06	0.37	1.8	0.08	1.3	0.24	0.98	1.3
	OH	H04DS_Oct	HVS3	Urban	Yes	Unknown	0.11	0.04	0.25	1.5	2.5	2.4	5.5	2	2.6	1.6	0.14	1.2	4.7	0.23	2.4	3.6	3.1	3.5
	OH	H04DS_Apr	HVS3	Urban	Yes	Unknown	0.11	0.026	0.27	1.3	1.8	1.6	1.6	1.4	2.2	0.016	0.01	0.22	3.9	0.17	1.6	0.044	2.4	2.8
	OH	H05aDS_Jun	HVS3	Urban	Yes	Unknown	0.05	0.02	0.12	0.7	3.9	0.25	2.6	0.72	1.1	0.21	0.05	0.21	2.2	0.09	0.91	0.01	1.1	1.5
	OH	H05SHD_Oct	HVS3	Urban	Yes	Unknown	1.6	0.29	2.70	17	18	16	38	11	20	1.8	0.01	3.9	46	2.8	12	17	33	34
	OH	H05SHD_Apr	HVS3	Urban	Yes	Unknown	1.9	0.27	3.30	14	17	13	33	13	21	1.6	0.046	2.7	45	2.6	16	0.34	32	32
	OH	H06DS_Jun	HVS3	Urban	Yes	Unknown	0.15	0.36	0.69	4.1	2	2.4	5.6	3.2	6.2	2.1	0.19	1.8	11	0.55	4.1	0.23	6.3	8
	OH	H06DS_Oct	HVS3	Urban	Yes	Unknown	0.17	0.19	0.33	3.9	3	4.4	9.8	2.7	3.2	0.64	0.19	0.69	4.9	0.36	2.8	1.6	4.5	5.4
	OH	H06DS_Apr	HVS3	Urban	Yes	Unknown	0.18	0.14	0.45	2.8	3.5	2.9	7	2.4	4	0.29	0.026	0.53	7.4	0.33	2.8	0.074	4.5	5.5
	OH	H07DN_Jun	HVS3	Urban	No	Unknown	0.24	0.1	1.10	5.1	7.7	6.6	15	5.3	7.2	1.9	0.23	2.1	15	0.46	6.9	0.06	7.5	11
	OH	H07DN_Oct	HVS3	Urban	No	Unknown	0.19	0.1	0.57	5	4.6	4.7	9.9	2.9	5.4	0.99	0.08	0.98	12	0.36	2.9	42	6	7.1
	OH	H07DN_Apr	HVS3	Urban	No	Unknown	0.33	0.11	1.20	6.4	9.1	7.1	17	6.4	9.8	0.88	0.036	1.3	20	0.56	7.5	0.048	10	14
	OH	H08D_Jun	HVS3	Urban	No	Unknown	1.1	0.52	3.90	24	54	41	103	35	34	7.2	0.36	7.5	90	2.1	41	0.35	41	68
	OH	H08D_Oct	HVS3	Urban	No	Unknown	1.8	0.34	5.80	40	41	37	108	22	43	3.5	0.06	9	88	3	27	12	43	69
	OH	H08D_Apr	HVS3	Urban	No	Unknown	1.1	0.24	3.50	20	23	17	42	16	27	2.1	0.056	3.4	48	1.7	19	0.2	26	37

[a] AZ: Arizona; IL: Illinois; IN: Indiana; IA: Iowa; KY: Kentucky; LA: Los Angeles; MA: Massachusetts; ME: Maine; MI: Michigan; MN: Minnesota; NJ: New Jersey; NY: New York; NC: North Carolina; OH: Ohio; PA: Pennsylvania; TX: Texas; WI: Wisconsin; WA: Washington.

[b] ACNP: acenaphthene; ACNPHT: acenaphthylene; ANTH: anthracene; BAA: benz[*a*]anthracene; BAP: benzo[*a*]pyrene; BEP: benzo[*e*]pyrene; BBKF: benzo[*b,k*]fluoranthene; BGHIP: benzo[*g,h,i*]perylene; CHRY: chysene; CORO: coronene; CPCDP: cyclopenta[*c,d*]pyrene; DBAHA: dibenz[*a,h*]anthracene; FLUORAN: fluoranthene; FLUOR: fluorine; I123CDP: indeno[1,2,3-*c,d*]pyrene; NAPH: naphthalene; PHEN: phenanthrene; PYR: pyrene.

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